Biomarkers for Alzheimer's Disease and Signs of Anxiety

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Abstract

Alzheimer's Disease (AD) and associated dementias have anxiety as a risk factor and prodromal symptom, although the underlying neurobiological underpinnings are yet unknown. The purpose of this systematic review and meta-analysis was to investigate the relationship between anxiety symptoms and amyloid-beta (A) and tau, two key indicators of AD neuropathology. Five databases were searched systematically for relevant literature. Studies examining the association between anxiety and tau and/or A neuropathology in cognitively healthy adults were included. Whenever possible, random-effects meta-analyses were used to integrate effect sizes across trials, individually for tau and A. Sensitivity studies were carried out to determine whether the results varied depending on the kind of anxiety (i.e., state and trait anxiety) and the modality used to measure the biomarkers (i.e., positron emission tomography and cerebrospinal fluid).

Keywords: Anxiety • Amyloid • Tau • Alzheimer's disease • Dementia • Neuropathology

Objective

By 2050, it is anticipated that the prevalence of dementia, of which Alzheimer's Disease (AD) is the most prevalent late-life type, would have tripled worldwide. Understanding modifiable dementia risk factors and underlying neurobiological correlates is crucial in tackling the anticipated rise in dementia cases because there are currently no effective treatment options. Of all mental health disorders, anxiety disorders have been found to be the most common. According to epidemiological studies, up to 33.7% of people in the West will have an anxiety disorder at some point in their lives, with subclinical anxiety symptoms believed to be twice as common as the full syndrome. Although anxiety is widespread in all stages of dementia, recent research suggests that it may also be a risk factor for developing dementia.

It is uncertain whether anxiety is a prodromal symptom or a risk factor for dementia. To enhance scientific knowledge of the connection between anxiety and dementia, however, it is essential to elucidate underlying neurological connections. Although this is a young field of study, conflicting results have been reported (for example, relationships between anxiety symptoms and AD neuropathology that are positive, negative, or absent). In order to address the confusion and clarify the connection between anxiety and AD neuropathology, a meta-analytic method is essential in this situation. Therefore, the current study's objective was to conduct a systematic review and meta-analysis to investigate the relationship between anxiety and two key amyloid-beta (A) and tau markers of AD neuropathology.

Terms associated with anxiety as well as those associated with A and tau were integrated for the database searches. Each database's search phrases were slightly modified to take into account the demands of various search engines, and in databases that permitted it, suitable MeSH terms were added to enhance the current search methodology. There were no restrictions on the date of publication, and animal studies were eliminated in accordance with Cochrane standards.

Screening was made easier with the help of Covidence. To find pertinent publications, two reviewers independently reviewed titles, abstracts, and complete texts against eligibility criteria. Discussions with a third reviewer helped to settle disputes in certain cases.

Studies were chosen if they met the following criteria: (i) they were cross-sectional (or, if longitudinal, included baseline analyses), (ii) they reported information on cognitively healthy adults with a mean age over 18 years, (iii) they assessed anxiety using a self-report symptom questionnaire or by using established clinical criteria (such as the International Classification of Diseases) for generalized anxiety disorder, (iv) they included an in vivo (such as positron emission tom. Studies that only included an informant-based assessment of anxiety (such as the Neuropsychiatric Inventory Questionnaire) or that primarily focused on participants with a significant medical or psychiatric disorder that was not generalized anxiety disorder were excluded. In cases where papers couldn't be found or further information was needed, the authors of the relevant research were contacted.

The following information was extracted from eligible studies using a standard form: (i) authors and year of publication; (ii) study sample characteristics; (iii) anxiety measurement; (iv) type and measurement of AD biomarkers; and (v) information needed for meta-analysis (such as correlation coefficients and sample sizes). Data were separately extracted by two reviewers, and any differences were addressed by a third reviewer after comparing the data extraction forms.

All studies used standardized self-report symptom measures to measure anxiety. Across research, eight different scales were used, with the State and Trait Anxiety Inventory being the most frequently used (k = 8; 29.6%). While the remaining studies analyzed only one form of anxiety, five (18.5%) examined both trait and state anxiety. Across investigations, the prevalence of self-reported anxiety was generally low (Supplementary Table 3). No research reported including participants with a clinical diagnosis of anxiety, while 14 studies (51.9%) specifically excluded participants with this condition. To assess the percentage of participants with anxiety symptoms who crossed the clinical threshold, nine research (33.3%) used known cut-off scores (median: 6.0%; range: 0.0% to 13.7%).

Although anxiety has been linked to a higher incidence of AD dementia, vascular dementia and all-cause dementia have shown greater and more persistent relationships. Beyond A and tau, AD dementia sometimes contains a complicated constellation of diseases. Taking into account our results in a broader perspective, it is likely that mechanisms other than an A or tau pathway are responsible for the link between anxiety symptoms and an elevated risk of dementia (including, indirectly, AD dementia).

The link between anxiety and AD dementia has been explained by a variety of possible etiologies. One putative biological mechanism that may be at play is the Hypothalamic-Pituitary-Adrenal (HPA) axis. Dysregulation of the HPA axis has been linked to anxiety disorders, and an over secretion of glucocorticoids is a clear result of this disruption. Increased glucocorticoids may make people more susceptible to dementia

by accelerating degenerative processes such hippocampus shrinkage. Furthermore, it is well recognized that high glucocorticoid levels raise the risk of cardiovascular and cerebrovascular conditions, which are in turn risk factors for Alzheimer's disease and vascular dementia. Another potential biological cause is inflammation. There is accumulating evidence that inflammation has a role in the pathophysiology of anxiety, especially early on in the AD continuum, before the buildup of A plaques. Systemic and intestinal inflammation have also been linked to the pathogenesis of anxiety. Another theory is that anxiety increases the chance of developing dementia by decreasing cognitive reserve (i.e., adaptability that explains how different aspects of cognitive function or daily functioning are affected differently by brain age, illness, or trauma). Indeed, older persons with clinically relevant anxiety symptoms have been found to have lower levels of cognitive reserve than those without these symptoms.

There were no correlations between anxiety symptoms and CSF t-tau or p-tau181 levels in three separate cohorts. Analyses that only included participants who reported anxiety symptoms above a cutoff suggesting a clinical level of anxiety (i.e., GAI 10 or GAI-short form 2) had the same results. Additionally, no correlations between anxiety symptoms and regional tau-PET standardised uptake value ratios (i.e., Braak stages I, III, and IV and the entorhinal cortex and inferior temporal cortex) were seen in two investigations that used the same cohort.

Conclusion

Conclusion: In cognitively healthy people, our meta-analytic syntheses of existing data showed no relationships between anxiety and AD neuropathology (i.e., A and tau). However, there may be a complex relationship between anxiety and dementia-related neurobiological correlates that goes beyond AD neuropathology and is supported by a number of other factors. Investigating the impact of many variables (such as the severity, chronicity, and timing of anxiety symptoms) that may act as mediators between anxiety and AD neuropathology will require large, life-course studies with thorough assessments. It is crucial for public health that we increase our knowledge of the neuropathological factors that connect anxiety and dementia since doing so could lead to the development of fresh ideas for preserving cognitive function as we age.

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